HYPOTHERMIA FOR CARDIOVASCULAR SURGERY: ACIDOSIS IN THE REWARMING PERIOD

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Blood gas changes and the related alterations in acid-base balance at lowered body temperatures have been extensively studied (Rosenhain and Penrod, 1951; Swan et al., 1953; McMillan et al., 1955; Axlerod and Bass, 1956). However, the changes associated with rewarming have evoked less interest, although most centres have encountered clinical problems during this period.

Metabolic acidosis has been reported in surface cooled hypothermic patients (Dill and Forbes, 1941) and in dogs, both during and after immersion hypothermia (Deterling et al., 1955). Brewin et al. (1955a, 1955b, 1955c, 1956) have published results of an intensive experimental study of metabolic acidosis, occurring in dogs rewarming after extracorporeal veno-venous cooling. They report evidence of metabolic acidosis found in patients recovering from cardiac surgery, after cooling by the same method.

Our experience has been outlined lately (Key, 1956), and a more detailed analysis of the biochemical findings is intended for the near future (Waddell et al., in press).

The purpose of this paper is to describe, in some detail, cases of acidosis encountered in clinical hypothermia and to comment on the anaesthetic and rewarming techniques employed. Using a standard anaesthetic technique, a high incidence of acidosis has been encountered in the rewarming period. On changing this technique, the incidence of acidosis fell markedly and it is proposed to present this material in the following manner:

1. Introduction.
2. A brief description of the original anaesthetic technique, giving details of each of the cases which became acidotic.
3. A brief description of the current anaesthetic technique, outlining those cases which have become acidotic.
4. A discussion of the factors involved, including the differential diagnosis of the causes of acute acidosis in the rewarming period.

INTRODUCTION

The problems to be presented are from a consecutive series of patients submitted to hypothermia.

Methods.

The technique for inducing hypothermia in this unit has been described (Bigelow et al., 1950, 1954), and consists essentially of surface cooling.

Blood sampling has proven technically difficult in cold patients. Arterial blood was preferred but, in a few instances, venous was accepted.

pH determinations have been performed on seventy-two consecutively drawn arterial and venous samples representing a pH range of 7.00 to 7.60 units and a temperature range from 38°C to 29°C. The venous value was never higher than the arterial and the difference was found to be greater at the alkalotic end of the range. Thus:

26 samples, giving values under 7.35 units (arith. mean 7.25 units), showed an average a-v difference of 0.026 pH units.
The patients were placed between Therm-o-rite blankets with ice bags applied to neck, axillae and groins and, in some cases, with crushed ice applied to trunk and thighs. Cooling was discontinued at a point where the estimated drift would achieve the desired temperature. The top blanket, ice bags and ice were removed, circulation of the refrigerant stopped and the operation started with the patient lying on the bottom cooling blanket.

Rewarming was often started during the operative period by circulating fluid, at 34°C to 38°C, through the bottom blanket. At the end of the procedure, the patient was dried, returned to bed with a few hot water bottles (at 38°C), and covered with two or three light warmed blankets.

An anticholinesterase was given in dosage sufficient to ensure adequate spontaneous respiration and, coincidentally, to promote shivering.

**Case Reports**

**Case 1. Mrs. N. C. Age 28.**

*Diagnosis.* Tetralogy of Fallot with cyanosis and left heart failure.

*Operation.* Open infundibular resection was performed at 29°C while circulation was interrupted for seven minutes.

*Rewarming.* Shivering was noted intermittently. The rewarming period was not remarkable until T_r reached 33.9°C.

\[ \text{pH after pre-operative value of } 7.30 \text{ (pH}_0 \text{ corrected value), the pH during cooling and operation ranged between 7.38 and 7.48 units. During the early rewarming period the pH declined gradually to 7.20.} \]

*Rewarming acidosis.* At T_r 33.9°C, the patient suddenly lost consciousness and ceased active movement, respiratory movements, hitherto considered perfectly adequate, were depressed, necessitating controlled respiration. No fall in blood pressure was noted, pH was 7.00 units. Decamethonium 3 mg was given to permit more vigorous hyperventilation, when the pH had fallen to 6.95 during 15 minutes' controlled respiration. After 15 minutes' hyperventilation, the pH had risen to 7.07 units and the patient soon regained consciousness, began to breathe spontaneously and move actively. The pH, after clinical recovery, was 7.12 units.

Observation was continued but no further complications were noted during rewarming. Respiratory excursions remained adequate and the pH showed a gradual rise.

*Remarks.* Serum sodium values, obtained for another study, were within normal limits throughout cooling, but on rewarming to T_r 37°C the level was 117 m.equiv/l.

This was the first case of severe acidosis detected in the rewarming period and its reversal was originally attributed to hyperventilation. It is now appreciated that muscular relaxation contributed to this reversal.

**Case 2. Mrs. F. D. Age 30.**

*Diagnosis.* A.s.d. and congestive heart failure.

*Operation.* Perauricular digital exploration of the right atrium and interventricular septum was carried out. The circulation was not interrupted since her lesion was not considered amenable to surgery. The lowest temperature reached was 28°C.

*Rewarming.* Rewarming was started during operation and continued between the Therm-o-rite blankets until the patient was transferred to bed at T_r 35.5°C. She was extubated, after breathing well without assistance for two hours, at T_r 34°C. Shivering was not
noted at any time. The postoperative course was smooth until the temperature was close to normal.

\[ pH \] The pre-anaesthetic level was 7.43. Subsequently, the pH rose to 7.55 during operation, then ranged gradually down to 7.26 at T_r 36.4°C.

Rewarming acidosis. At T_r 36.6°C, the pH was 7.12 units and respiration was assisted for 15 minutes before the patient lost consciousness, at pH 7.03. Associated with the loss of consciousness were loss of virtually all respiratory activity, cessation of active movement, fall in systolic blood pressure and a rise in heart rate.

Despite vigorous hyperventilation with muscle relaxant, 1,000 ml of citrated blood and 5 grams of NaHCO_3, the pH continued to fall and consciousness and respiration remained depressed. Blood pressure was partially restored, temporarily, but systolic levels were not raised above 80 mm Hg. The tachycardia persisted and, after increasingly frequent ventricular premature beats, irreversible ventricular fibrillation supervened. The last pH obtained before death was 6.80 units.

Remarks. Acidosis must be implicated in this woman's death although severe heart disease was certainly contributory and blood replacement may have been somewhat inadequate.

The sodium level during the profound acidosis—and prior to the NaHCO_3 infusion—was 125 m.equiv/l. Previous values were within normal limits.


Diagnosis. Tetralogy of Fallot with cyanosis and left heart failure.

Operation. A Blalock procedure was performed, without incident, at T_r 31°C.

Rewarming. Rewarming was entirely spontaneous and was uneventful until the temperature had risen to T_r 36°C, when the patient was fully conscious.

pH. The pre-anaesthetic level was 7.39. A rise to 7.63 was noted during cooling and operation. During early rewarming the pH declined to the "control" level.

Rewarming acidosis. At T_r 36°C, the pH was 7.19 units. Decamethonium 3 mg was given intravenously and vigorous hyperventilation was started. Within five minutes, consciousness was lost. There was associated hypotension and tachycardia and it was noted that the pH had fallen to 7.13 units.

Hyperventilation was continued and a 1 per cent NaHCO_3 infusion started at a rapid rate. Improved level of consciousness was noted within ten minutes and the pH had risen to 7.20 units. The rate of NaHCO_3 infusion was slowed.

At T_r 37.5°C, the pH was 7.29 units. 2.5 grams of NaHCO_3 had been given. The patient was again fully conscious, breathing well, reacting to the endotracheal tube and moving actively. His condition remained good and the pH showed a slight further rise.

Remarks. Depression of consciousness in this case was apparently due to a moderate acidosis. Clinical recovery seemed related to the NaHCO_3 infusion.

Case 4. Mr. H. L. Age 22.

Diagnosis. Aortopulmonary window with cyanosis and congestive failure. Possible septal defect.

Operation. Postauricular digital exploration revealed a v.s.d., which was considered inoperable under hypothermia. The lowest temperature reached was 30°C.

Rewarming. The bottom blanket was warmed during operation but external heating was stopped at T_r 32.3°C, when the patient was transferred to bed. Rewarming was complicated by unusual ventricular irritability. Attempted hyperventilation was associated (with the thorax closed) with frequent ventricular extrasystoles and could not be maintained. Procaine amide, intravenously, did not reduce cardiac irritability. Gross shivering was noted.

pH. After a pre-anaesthetic value of 7.33 units, the pH varied between 7.44 and 7.56 units during cooling and operation, then fell to 7.24 units during rewarming.

Rewarming acidosis. At T_r 34°C, NaHCO_3 5 per cent was given, intravenously, in a 50 ml dose, because the pH had declined to 7.24 and cardiac irritability prevented hyperventilation. This produced elevation of the heart rate and blood pressure without changing the pH trend. The pH had fallen to 7.03 units by the time the temperature had risen to 36.2°C. A 2.5 per cent NaHCO_3 infusion was started at a rate of 5 ml/min. After twenty-five minutes the pH had remained at 7.03 units and the infusion rate was slowed a little. Gross shivering recurred and chlorpromazine 25 mg was given, intravenously. No clinical signs of acidosis appeared until T_r 37.5°C, when definite depression of consciousness, respiration and active movement was noted. The pH was 6.95 units. Respiration was assisted and the rate of NaHCO_3 infusion increased.

After 25 grams of NaHCO_3 had been given, the patient suddenly regained consciousness and once again began to breathe spontaneously and move actively, at 37.7°C. The pH was 7.18 units. Eventually, the pH stabilized a little above 7.30 and no further deviations were noted.

Remarks. The slow response of acidosis to a large dose of NaHCO_3 was impressive. The amount given raised the serum sodium from 146 to 148.4 m.equiv/l. The patient did not develop oedema subsequently, although an easily controlled episode of acute nocturnal dyspnoea occurred on the third postoperative night.

Comments

In the four cases of severe acidosis, sudden loss of consciousness, in hitherto conscious patients, was the most striking feature. When this occurred, pH values were found to be between 7.00 and 7.13 units.

In the few instances when CO_2 combining power estimations were made during periods of acidosis,
low values demonstrated a predominantly metabolic element in the acidosis.

Attendant upon loss of consciousness, with a falling pH, was generalized muscular weakness in patients who had previously been moving actively and breathing spontaneously and fully. With the muscular weakness, respiration became obviously inadequate and required controlled respiration in each case.

The blood pressure fell and the heart rate rose in two patients, who had not received NaHCO₃, at the time of onset of symptoms.

Finally, correction of the acidosis was associated with return of consciousness, resumption of adequate spontaneous respiration and active movements and with a decline in heart rate and restoration of blood pressure—this being almost as dramatic as the onset of this condition. When recovery occurred, pH values were found to be between 7.12 and 7.20 units.

The measures instituted to correct the acidosis reflect one's thinking at the time these problems were encountered.

In Case 1 respiratory acidosis was immediately suspected and hyperventilation started with a muscle relaxant. In retrospect it is probable that both hyperventilation and relaxation contributed to the correction of the pH.

Case 2 proved resistant to attempted hyperventilation (without a relaxant) and a small dose of bicarbonate.

Sodium values in the period of profound acidosis, in the first two cases, were of reassurance in the use of sodium bicarbonate in the treatment of the third case. The rapid restoration of consciousness, after a small dose of bicarbonate, was impressive, although the dosage used would not influence the pH greatly.

In Case 4 hyperventilation could not be achieved and correction of the acidosis must be attributed to the large dose of NaHCO₃ used in conjunction with medication to control shivering.

pH (venous values as black dots) has been plotted against rectal temperature in ten cases (fig. 1). The median curve is representative of the individual tendency in each case with some rise in pH during cooling and a fall during rewarming.

Four cases of severe acidosis, in ten cases studied, and an overall incidence of acidosis in 50 per cent of cases prompted revision of the anaesthetic and rewarming technique.

GROUP B
The aims, in the attempt to prevent acidosis during rewarming, were several fold:

(1) Maintenance of peripheral circulation throughout hypothermia.
(2) Control of respiration, during cooling and operation, in such a way as to keep the blood pH within the normal range.
(3) Abolition of shivering.
(4) Promotion of adequate spontaneous respiration as early in the rewarming period as possible.

The current technique is described elsewhere (Fairley, 1957), but, in summary, is as follows. The patient is sedated, on the night before operation, with oral promethazine. Promethazine and chlorpromazine are given intramuscularly one hour pre-operatively. Induction of anaesthesia is achieved with an intravenous infusion of promethazine, Chlorpromazine and pethidine, administered over 15–30 minutes. This is usually sufficient to abolish the eyelash reflex, but has not proved an embarrassment in patients with severe heart disease. Relaxation is provided, for intubation, by suxamethonium and these patients are ventilated with nitrous oxide-oxygen, by the Jefferson ventilator, using d-tubocurarine intermittently. Control of a rising pH is achieved by taking the soda lime out of the circuit and, in some cases, by reducing the flow of gases.

The cooling technique is identical to that previously employed.

Shivering during rewarming is controlled by intravenous or intramuscular chlorpromazine.

Spontaneous respiration during rewarming is promoted by the use of an anticholinesterase, whenever indicated.

It was originally planned to rewarm the patient, when possible, by the use of warm fluid circulated through the blankets and by hot water bottles. However, many of the second group of patients had no more active measures for rewarming than the former.
**pH vs. RECTAL TEMPERATURE**

- **10 patients**

![Graph showing pH vs. Rectal Temperature](#)

**Case Reports**

**Case 5.** Master C. B. Age 16.

*Diagnosis.* Fallot's tetralogy with cyanosis and congestive heart failure.

*Operation.* Transventricular infundibular resection was performed, at Tr 29°C, with seven minutes' circulatory occlusion. Ventricular fibrillation followed cardiac massage, instituted for poor heart action, after the circulation was restored.

*Rewarming.* Rewarming was initiated during surgery, by warming the bottom blanket, and was continued between the Therm-o-rite blankets until the patient was transferred to bed, at Tr 31.7°C. No further external heating was used because of moderate hypotension. The patient was extubated at Tr 32.5°C, when reacting violently to the endotracheal tube. Shivering was not noted. He was fully conscious, talking rationally, breathing adequately and moving well at 33°C. Heart rate and blood pressure were satisfactory at this point.

Shortly after reaching Tr 33°C, he became hypotensive and plasma expander was given, while awaiting more whole blood. The level of consciousness declined gradually and manual control of respiration was started. The blood pressure could not be maintained despite further blood and plasma expander. He developed cardiac arrest at Tr 34.5°C. The chest was opened but attempted resuscitation was not successful.

*pH.* The pre-anaesthetic pH was 7.44, and subsequent samples within normal limits. The first postoperative samples were 7.31 and 7.28 and chlorpromazine 50 mg was given, despite the absence of shivering. Prior to the depression of consciousness, the pH declined to 7.24 units.

*Rewarming acidosis.* Significant acidosis was found at 33.4°C, when the pH was 7.19 units.

*Remarks.* This case was in acidosis due to oligemic shock and, in retrospect, chlorpromazine was not indicated.

*pH* values below 7.25 have been found in two other cases:

**Case 6.** A 25-year-old woman, severely disabled with an a.s.d. and pulmonary stenosis, developed a moderate acidosis—with no clinical manifestations—after circulatory interruption of four minutes, for open pulmonary valvotomy at Tr 29.2°C. She had...
four episodes of ventricular fibrillation, lasting between two and ten minutes, but sinus rhythm was eventually restored. Rewarming was uncomplicated, despite a pH reading of 7.18, at 31.2°C. There were no clinical signs of acidosis and the pH rose without measures to correct the imbalance.

Case 7. Finally, several values below 7.25 were obtained (7.17 – 7.23) during rewarming in a patient in whom the complicating factor was minimal residual curarization (in an abdominal aortectomy, with very low urinary output). This patient was semicomatose for a prolonged period but did not show the sudden loss of consciousness associated with severe acidosis in the earlier cases. Neostigmine and atropine had been given at the time of finishing the operation but the acidosis was not reversed until a further dose was given, some time later, with subsequent spontaneous hyperventilation. He made an uneventful recovery.

Comments
The pH values have been plotted against the rectal temperature, at the time of sampling, in 19 patients (fig. 2). Again, the median represents the usual tendency with some rise during cooling and a gradual decline during rewarming. In many instances this is followed by a second rise, giving a sinusoidal curve, and often rising above the pre-operative value.

A pH below 7.14 has not been observed, in the second group of patients, nor has the "acute acidotic syndrome of rewarming" been encountered.

Rewarming rates. Several variables were introduced with the modifications described, but it is instructive to compare the rewarming rates in the two groups. The rewarming rate of the first group, expressed in degrees centigrade/hour/70 kg body weight, ranged from 0.47°C to 3.49°C. The arithmetic mean was 1.82°C and the standard deviation 0.98. The range in the second group was 0.78°C to 1.99°C/hour/70 kg. The arithmetic
mean was 1.34°C and the standard deviation 0.31. The difference between the means is significant at the 5 per cent level. This may be attributed to depression of shivering (in group B). The use of chlorpromazine is an important factor in the control of shivering but somewhat greater use of external heat may have been a contributory factor.

DISCUSSION

Without elaborating on the many factors involved in the maintenance of acid-base balance, it is evident that in hypothermia by surface cooling, using controlled respiration, the following influences exist:

(1) Lack of spontaneous respiratory compensatory mechanisms.

(2) Peripheral vasoconstriction due to cold and to respiratory alkalosis, should the latter be allowed to occur.

(3) Shivering and increased muscle tone, with resulting rise in lactates and other acid metabolites in the blood.

(4) Probable depression of renal control of hydrogen ion concentration.

The cases in group A would appear to have been exposed to these influences, since, with an anaesthetic technique commonly used in thoracic surgery, a high incidence of acidosis was encountered in the rewarming period. Reference to the results of the Toronto General Hospital neurosurgical team (also using surface cooling) showed that their anaesthetic technique for hypothermia (Vandewater et al., 1955) did not produce a rewarming acidosis (Vandewater, 1957). By the use of (1) spontaneous respiration, (2) antishivering agents, and (3) peripheral vasodilators, they were able to circumvent respiratory alkalosis and metabolic acidosis, to a large extent. They commonly observe a slight rise in pH during cooling and a fall of similar proportions during rewarming.

Alkalosis. There are several theoretical objections to alkalosis during cooling, viz., shift of the oxygen dissociation curve to the left, peripheral vasoconstriction and, possibly, renal discard of sodium bicarbonate, etc. An attempt has been made to keep the blood pH within normal limits in group B. The methods used have been mentioned. Figure 2 shows fewer readings above 7.45 and demonstrates some measure of success.

Chlorpromazine. This agent offers practical and theoretical advantages in facilitating cooling (Dundee et al., 1954), inhibiting shivering and maintaining peripheral circulation. Peripheral vasoconstriction is considered deleterious in that it may foster peripheral hypoxia, with consequent promotion of anaerobic glycolysis and increased lactate production. This is a similar mechanism to that producing lactate accumulation in oligaemic shock (Wiggers, 1950). Chlorpromazine is used to prevent gross shivering during the rewarming period, in an attempt to prevent the sudden massive production of lactates and other acid metabolites of muscular catabolism. Depression of shivering maintains anion production within limits which can more readily be controlled, by the compensatory hyperventilation of the spontaneously breathing postoperative patient, provided there is no gross pulmonary pathology present.

The simultaneous introduction of more than one variable, in the more recent anaesthetic technique, prevents any comment as to the relative importance of the various factors operative in this method. However, it is believed that the prophylaxis of this technique, against the acute acidotic syndrome of rewarming, is to be preferred to the necessity of undertaking the active therapy of this condition.

Differential diagnosis of rewarming acidosis.

From the discussion of the factors involved, it is evident that a low blood pH, in the rewarming period, could be due to any of several factors:

(1) Metabolic acidosis. This has been discussed above the cases 1–4 are representative.

(2) Respiratory acidosis. The similarity between the metabolic acidosis of rewarming, described above, and the respiratory acidotic syndrome reported by Scurr (1954) is remarkable. Adequate spontaneous respiration, in the rewarming period, is invaluable, as a means of combating impending metabolic acidosis, and it should be noted that, following the use of relaxant agents, ventilatory power which normally would be adequate may be incapable of the hyperventilation necessary for this compensation. This was seen in case 7.

(3) Haemorrhage. The low pH values obtained in haemorrhagic shock are well recognized (Bland,
and may cause differential diagnostic confusion in cardiovascular surgery. This was seen in case 5. Massive transfusion, with resulting increase in citrate radicles, will contribute to the acidosis (Brewin and Neil, 1954; Yendt, 1957).

(4) Peripheral embolism. One has seen a case of gross metabolic acidosis, resistant to therapy, occurring in a patient who sustained multiple peripheral emboli (including an aortic saddle embolus), when an atrial myxoma was encountered unexpectedly. This was presumably due, in part, to peripheral anoxic metabolism.

Treatment of the acute acidotic syndrome.

Some decline of the blood pH level, in the rewarming period, is to be expected in the majority of cases. However, this level rarely falls below 7.30 units. Should readings continue to drop and signs of the acute acidotic syndrome develop, this should be considered an emergency situation.

From the discussion of the differential diagnosis, it will be evident that more than one factor may be contributing to the acidosis. Thus, although accumulation of acid metabolites is the probable cause of the falling pH, consideration should be given to the respiratory exchange and to blood volume, etc. Whatever the cause of the acidosis, a muscle relaxant will permit hyperventilation through soda lime and the resulting reduction in plasma CO₂ will temporarily reverse the falling pH level.

If shivering has occurred, chlorpromazine 25–50 mg should be given intravenously and, in extreme cases, an intravenous infusion of sodium bicarbonate should be started. Careful and frequent observations of blood pH levels should be used as the clinical yardstick, as to the efficacy of the therapy; in addition, total blood CO₂ or alveolar CO₂ levels may be of assistance. It is felt that while dosage of bicarbonate may be calculated from widely recognized formulae, the latter were designed for the treatment of diabetic ketoadidosis, which is probably not analogous to the complex circumstances of recovery from cardiovascular surgery under hypothermia. Therefore, it is safer to adjust the dosage according to the blood pH response.

Any treatment of an acute metabolic acidotic crisis is aimed at tiding the patient over his temporary inability to maintain a normal H-ion concentration, in the presence of gross lactate accumulation.

It is of interest to note that the acute acidic syndrome occurred in patients who had neared normal temperature. Because of this, temperature should not be used as a yardstick of fitness for return to the ward. Experience has shown that a rising pH is the best index of the patient’s well-being in the rewarming period.

In retrospect, cases of sudden death during rewarming, or others exhibiting a nonspecific picture of hypotension and impaired consciousness, prior to restoration of normal body temperature, may have been acidic. It is possible that the occasionally described, rather vague conditions, “rewarming shock” and “rewarming death” may be explicable on a basis of metabolic acidosis.

SUMMARY

A report has been made of a series of cases in which severe metabolic acidosis arose, during the rewarming period following hypothermia for cardiac surgery. A further series is reported, showing that this syndrome is not encountered when care is taken to avoid (a) shivering, (b) peripheral vasoconstriction, (c) respiratory alkalosis during cooling and operation, and (d) when spontaneous respiration is established at the earliest opportunity, postoperatively.

The differential diagnosis of the causes of severe acidosis in the rewarming period and the treatment of the acute acidotic syndrome are discussed.

ACKNOWLEDGMENTS

The authors would like to express their indebtedness to Mrs. J. Woodhouse, B.Sc., whose technical assistance in the estimation of blood pH levels has been invaluable. Her work has been supported by a grant from the Defence Research Board.

REFERENCES


**BOOK REVIEW**


This new edition of W. G. Holdsworth's book on cleft palate and hare lip surgery is a further improvement on the excellent first edition. The chapter on development by Professor Richard Harrison makes interesting reading, but the parts which are, of course, of most importance to anaesthetists are the three chapters on pre-operative management of the infant, on general anaesthesia for these cases, and on postoperative care. The chapter on general anaesthesia is, as before, by Drs. George Ennis and D. A. Sherman, and they are to be congratulated on the clear and concise way in which they describe their technique. There is no doubt that the special experience, both surgical and anaesthetic, that is acquired in the plastic surgery units has contributed to the greatly lowered morbidity and mortality of lip and palate surgery. There is much to be said for and against segregation of special cases into special units, but the writer feels that there is an unanswerable argument for allowing these babies to receive the full benefit of the techniques developed and practised in the plastic surgery hospitals (and in a few paediatric hospitals). The anaesthetist will find much of importance and interest in the chapters on pre- and postoperative assessment and care. Professor T. P. Kilner and the late Dr. John Hunter performed many thousands of operations and anaesthetics for hare lip and cleft palate without a death and this result was partly achieved by the co-operation as a team of the surgeon, anaes-

**BRITISH JOURNAL OF ANAESTHESIA**